hampered by the absence of controls receiving placebo treatment. When participation requires a high incidence of cystitis in the preceding year historical controls are bound to be biased. Not impressed by earlier work, leading textbooks on antibiotic treatment question the use of methenamine,[3 4] and we were therefore surprised when the code was broken and the efficacy of the drug shown.

We agree that conventional antibiotics may be even more effective for some time, but, as stated in the letter, they may promote bacterial resistance and hypersensitivity reactions.

Though methenamine has been used for almost a century, our investigation seems to be the first randomised, double blind, placebo controlled, long term crossover study ever performed with this drug to test its preventive effect in patients with recurrent acute cystitis. As it is well tolerated and effective and fails to produce cross resistance to conventional antibiotics it should remain a suitable prophylactic agent for women not only in Scandinavia but also in the United Kingdom.

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Converting enzyme inhibition and kidney function in normotensive diabetic patients with persistent microalbuminuria

SIR,—We have observed the effects of converting enzyme inhibition on renal function in hypertensive, insulin dependent diabetic patients to be much less dramatic than those observed by Dr Michael Marre and colleagues in normotensive patients (6 June, p 1448).

We studied eight insulin dependent diabetic patients with newly diagnosed hypertension (systolic or diastolic pressures greater than the 95th centile norm for age and sex) but without established nephropathy (urinary protein excretion <200 mg/day) during a six week run in period followed by a six week course of captopril 12·5 mg three times a day. All eight (five men, three women, mean (SD) age 50 (12) years) complied fully with treatment, as assessed by counting the number of tablets taken.

Supine and standing blood pressures were measured by random zero sphygmomanometer (Hawksley, England), urinary albumin excretion rate from a 24 hour collection by an enzyme linked immunosorbent assay (ELISA), and glomerular filtration rate and effective renal plasma flow by a double isotope technique and serial blood sampling with ^{51}Cr-edetic acid and ^{125}I-orthoiodohippurate, respectively. Supine and standing plasma renin activities were measured by radioimmunoassay (normal: supine 1·17-2·39 pmol/ml/hour; erect 2·99-4·30 pmol/ml/hour), as were supine and standing aldosterone concentrations (normal ranges: supine 100-500 nmol/l; standing 150-1500 nmol/l). Renal vascular resistance was determined by dividing the average of supine and erect mean blood pressures by effective renal plasma flow and the free filtration fraction by dividing glomerular filtration rate by effective renal plasma flow.

While we found an expected improvement in mean blood pressure, particularly when patients were standing, we found no significant changes in albumin excretion rate, renal haemodynamics, plasma renin activity, or aldosterone concentrations (table). This may relate to the relative excess of inactive renin produced in hypertensive diabetics, particularly those with autonomic neuropathy,[1] which in turn is often associated with disturbed renal function in insulin dependent diabetics.[2]

The observation by Dr Marre and coworkers that the glomerular filtration rate rose when patients received enalapril is particularly interesting in that while the median albuminuria excretion rate fell overall, it rose in three patients during treatment with enalapril. As glomerular hyperfiltration may be linked to the development and progression of diabetic nephropathy,[3] it is important to know whether glomerular hyperfiltration was a feature in these three patients despite a presumptive cumulative effect of reduced systemic blood pressure on regional intrarenal haemodynamics suggested by reduced renal vascular resistance. It might thereby be argued that converting enzyme inhibitors may have a detrimental effect on renal function in some diabetic patients.

Though the study by Dr Marre and colleagues differed from ours with regard to selection of patients and duration, we believe that a beneficial effect of converting enzyme inhibitors on renal function in hypertensive, insulin dependent diabetic patients without established proteinuria remains to be proved.

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Median (range) albuminuria excretion rate, and mean (SEM) estimates of renal haemodynamics, plasma renin activity, and aldosterone concentrations in eight hypertensive insulin dependent diabetic patients

	Initially	During six week run in period	During six weeks of treatment with captopril 12·5 mg three times a day
Albuminuria excretion rate (μg/min)	15·3 (7·1-53·3)	9·8 (1·5-19·8)	7·4 (2·0-46·2)
Glomerular filtration rate (ml/min/1·73 m^2)	129 (8)	125 (8)	123 (6)
Effective renal plasma flow (ml/min/1·73 m^2)	509 (40)	490 (25)	492 (22)
Free filtration fraction	0·26 (0·01)	0·25 (0·01)	0·25 (0·01)
Renal vascular resistance (mm Hg/ml/min)	0·24 (0·02)	0·25 (0·01)	0·23 (0·01)
Supine plasma renin activity (pmol/ml/h)	1·67 (0·11)	2·05 (0·17)	2·13 (0·33)
Standing plasma renin activity (pmol/ml/h)	2·85 (0·30)	3·29 (0·28)	3·46 (0·53)
Supine aldosterone concentration (nmol/l)	211 (29)	164 (18)	222 (55)
Standing aldosterone concentration (nmol/l)	358 (56)	300 (32)	374 (69)

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Loin pain and haematuria syndrome

SIR,—Though interesting, the observations of a possible association between the loin pain haematuria syndrome and intrarenal arterial spasm by Dr V Bergroth and colleagues (27 June, p 1657) must be interpreted with caution.

This is a poorly understood condition for which specific diagnostic criteria are lacking. The diagnosis is even more difficult because some patients have frank neurotic features.[1 2] It is important to guard against the diagnosis being inappropriately attached to a miscellany of organic and functional conditions. In our experience haematuria of renal origin, as determined by phase contrast urine microscopy,[3] is a feature of the syndrome, and it is disappointing that this was omitted from the report by Dr Bergroth and coworkers. In addition, C3 deposition may occur in various conditions, but in patients with the loin pain haematuria syndrome it is usually abundant and seen consistently in arteriolar walls, although occasional scanty deposits may be seen in glomeruli. Other immunological reactions are usually absent.

As arterial spasm during angiography may be caused by technical factors[4] it would have been appropriate for Dr Bergroth and colleagues to include a brief description of their angiographic technique. In addition, as the authors are suggesting that the mechanism for loin pain in this syndrome is renal cortical ischaemia caused by arterial spasm mediated by the autonomic nervous system, it would have been interesting to know if their patients experienced pain while arterial spasm was occurring.

The renal cortical defects described by Spriggs and Brantley are similar to some of those seen in the loin pain haematuria syndrome,[2 5] but it is difficult to see how transient spasm might account for the more advanced changes that have been described.[5 6]

A more carefully detailed description of intrarenal arterial spasm is required before it can be accepted as an underlying mechanism in this syndrome.

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6 Burden RP, Booth LJ, Ockenden BG, Boyd WN, Higgins P McR, Aber GM. Intrarenal vascular changes in adult patients with recurrent haematuria and loin pain—a clinical histological and angiographic study. *Q J Med* 1975;44:433-47.

AUTHORS' REPLY,—We agree with Dr Hutchison's general statement that it is important to guard against the diagnosis being inappropriately attached to a miscellany of organic and functional conditions. He himself, however, seems to belong to the "believers": those who believe that the loin